CHAPTER II

SEASONAL GRAVIMETRIC CHANGES IN THE ORGANS OF NORMAL AND ADRENAL MANIPULATED FERAL BLUE ROCK PIGEONS, <u>COLUMBA LIVIA</u>

Adrenal glands have attracted the attention of scientists since the early phase of 19th century. However, most of the earlier works dealt with histomorphological aspects in different species of birds and mammals (Findlay, 1920; Riddle, 1923; Muller, 1929). These studies were later extended to seasonal alterations in adrenal histomorphology (see Höhn, 1947; Kornfeld, 1957; Legait and Legait, 1959). Subsequently, further studies carried out on adrenals revealed several aspects of the functional involvement of adrenal steroids. The first definitive study of steroid secretion of avain adrenal gland was made by Phillips and Chester Jones (1957) who showed that adrenal effluent plasma from Capons contained predominantly corticosteroid with much smaller amounts of Cortisol, Cortisone and aldosterone.

The involvement of corticosteroids in several aspects of tissue functions is gaining interest in recent years. Of late, adrenal-gonad axis has caught the attention of scientists and is being studied in many species. Cyclic changes in the adrenals alongwith gonadal cyclicity has been reported to occur in fishes, repitles, birds as well as in mammals (Moens and Coessens, 1970; Datta <u>et al.</u>, 1978; Pankakoski and Klaus, 1982; Patel <u>et al.</u>, 1985; Ramachandran and Patel, 1986).

Both antagonistic as well as synergestic interactions between adrenal steroids and testicular secretions have been reported in birds and mammals (Boas, 1958; Soule and Assenmacher, 1966; Daniel and Assenmacher, 1969; Assenmacher and Boissin, 1970; Bengt, 1975; Datta <u>et al.</u>, 1978; Chaturvedi and Thapliyal, 1980, 1981). In majority of the cases, a definite involvement of adrenal principle(s) in reproductive activities has been noted. There are conflicting reports regarding the involvement of adrenals in reproductive physiology since, age, sex, sexual maturity, pituitary activity at the time of experiment, photoperiod and species, all significantly affect the type of interactions.

It is these conflicting and contradictory views regarding the adrenal-gonad relationship in animals that prompted the present study on blue rock pigeons. Apart from the adrenals, the thyroid has also been reported to undergo season specific alterations with respect to gonadal cycles. As thyroid is now known to play an important role in the reproductive physiology of birds, gravimetric study of the thyroid gland on a seasonal basis in normal and experimental birds was also undertaken.

Uropygium, a characteristic sub-cutaneous gland of birds has been shown to undergo season specific alterations in its absolute and relative weights (Patel and Ramachandran, 1983) and has also been proved to be a target organ for gonadal steroids (Deadhikari and Bhattacharyya, 1982; Gupta and Maiti, 1983). Further, in recent years, androgen receptors in preen gland of male Japanese quail have been characterized by Amet <u>et al.</u> (1982). Circulating levels of gonadal steroids are known to undergo seasonal changes and as adrenal steroids are known to have some effect either directly or indirectly on gonads, gravimetric studies of the uropygial gland on a seasonal basis in normal and experimental birds were undertaken.

Spleen has also been known to undergo alterations in its absolute or relative weight in both wild as well as domestic pigeons, <u>Columba livia</u> (Patel 1982; Patel and Ramachandran, 1983). Alterations in spleen weight with respect to adrenal weight have been reported in bats (Jonathan, 1980); the adrenal- spleen axis being a parallel one till adulthood is reached. Endogenous circulating level of corticosteroids is known to affect the red pulp/ white pulp ratio in certain strains of mice with adrenalectomy resulting in increased red pulp content₀ (Semenkov and Afinogenova, 1982). To have a general idea regarding the adrenal-spleen interrelation in pigeons, the weight of spleen in normal and experimental birds was recorded during the breeding and non-breeding phases.

MATERIALS AND METHODS

As described in Chapter I

RESULTS

All the organs studied exhibited season specific alterations in their weights, with both absolute and relative weights exhibiting similar pattern of changes (Tables 1 - 5).

Changes in Normal Birds

Adrenals exhibited a progressive increase in absolute weight from a minimum during regression through recrudescence to breeding as revealed by the steady increase in its total weight equivalent to 82% on the whole. This is further confirmed by the histological studies which showed increased activity along with the activity of gonads (Chapter III). Relative weight differences were found to be more marked from recrudescence to breeding than from regression to recrudescence.

Gonads as expected were found to weigh least during the quiescent phase (Nonbreeding months). Both the ovaries and testes were found to increase in weight(absolute weight) when the birds entered the preparatary phase (Recrudescence). The increase in weight being more dramatic in the case of testes than ovaries which on a percentage basis was 148% in the case of testes as opposed to only 27% in the case of ovaries. However, a marginal fall of about 24 % and 14% respectively in absolute weights occurred between recrudescence and breeding. Regarding relative weights, the pattern of changes was more or less similar as that of the absolute weights.

In contrast to adrenals, the thyroid revealed an altogether different pattern in the sense that there was notable reduction in its weight with increasing activity of the gonads. The reduction on a percentage basis being equivalent to 48% and 15% from regression to recrudescence and from recrudescence to breeding respectively. These gravimetric changes are well supported by the histological observations (Chapter III). Spleen weighed maximum during the regression phase and least during the breeding phase with an intermediate weight during the recrudescent phase. On a percentage basis the decrement in weight was to the tune of 30%.

Uropygial gland recorded an increase in weight during the breeding phase and the percentage increase from regression to breeding was about 25%.

| REPRODUCTIVE PHASES RECRUDESCENT | | | | | | | | | |
|--|---------------|-----------------|---------------|-------------------|-----------------|--------------------|-------------------|--------------------|---------------|
| PHASES RECRUDESCENT | NORWAL | | DEXAMETHASONE | E | ACTH | | CORTIC | CORTICOSTERONE | |
| RECRUDESCENT | | Boug | 120ug | 160ug | 0.5 I.U. | JugM | 1ugE | JugM | 3ugE, |
| | | 3 | 3 | | | | | | |
| A. W. | 17.26 | 14.01 | 11.65*** | 10.10 | ŧ | ł | ł | 1 | 1 |
| | ±1. 94 | ±1.78 | ±1.39 | ±1.25 | | | | | |
| R.W. | 6.317 | 5.67 | 4.83* | 4.56 ⁰ | I | ı | I | I | ı |
| | ±1.01 | ±1.92 | +1.01 | <u>+</u> 1.00 | | | | | |
| BREEDING A.W. | 23.87 | 13.30*** | 11.82 | 10.15*** | I | 1 | ı | ı | ı |
| | +3.01 | ±1•47 | •• | | | | | | |
| R.W. | 9.50 | ישי ** * | 5.08 | 4•43 *** | | | | | |
| | <u>+</u> 1.00 | ±0.50 | 10.12 | 10.45 | | | | | |
| REGRESSION A.W. | 15.97 | ı | ł | · | 23.42** | 18.80 ⁰ | 22.76 | 21.00 ⁰ | 22.90 |
| | <u>+</u> 2.57 | | | | +3.42 | +3.10 | +1.91 | т | 2.75 |
| R.W. | 6.02 | I | I | ł | ** 00°6 | 7.35 ⁰ | 6.62 | 8.26 ⁰ | 9 • 58 |
| | + 0•69 | | | | +0.93 | +0.76 | 41.11 | <u>+</u> 0.82 | ±1.40 |
| | @ P 0.02 | д + | 0.01 * P | 0•05 ** I | P 0.005 | 0 d *** | 0•0005 | | |
| | M - MORNING | । मि | EVENING A | A.W ABSOI | ABSOLUTE WEIGHT | П. К. М. – | - RELATIVE WEIGHT | WEIGHT | |

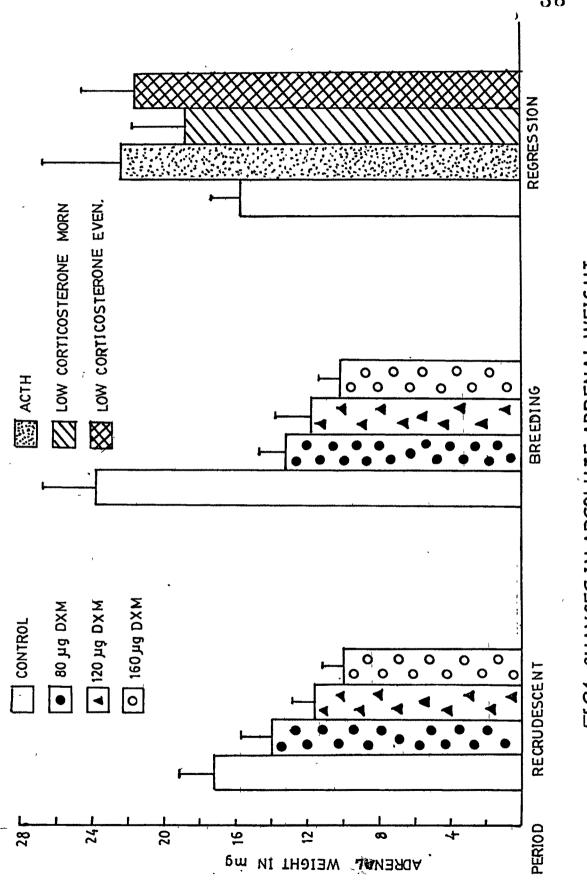
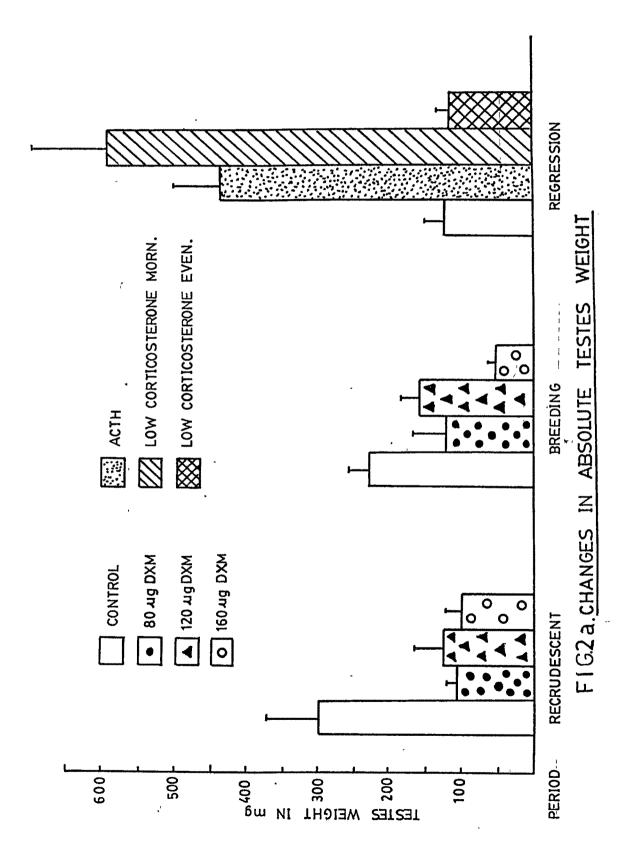


FIG1. CHANGES IN ABSOLUTE ADRENAL WEIGHT

| REPRODUCTIVE PHASES | NORMAL | Boug | DEXAMETHASONE 120µg 16 | SONE 160µg | ACTH 0.5 I.U. | Mault. | 1JJRE CORT | CORTICOSTERONE Jugh | ZµgE |
|------------------------|------------------|-----------------------|---------------------------|----------------------------------|------------------|--|--------------|------------------------|---|
| RECRUDESCENT | ŕ | | | | | | | | |
| A.W. | 301.54 | 101.28* | ** 127.48 | 101.28 *** 127, 48 *** 97.78 *** | 1 | ł | 1 | 1 | ı |
| | 460.25 | 420.09 | 43 | •77 ±20.60 | | | | | |
| R.W. | 109.65 | **** | ** 49 •35 | 49.35 <u>***</u> 40.60 *** | | | r | | |
| | +18.0 | <u>+</u> 8.90 | 49.90 | <u>+</u> 10.00 | | | | | |
| BREEDING | 70 LCC | マリマ **** ひり リァマ | | **** 0 0 | ł | ł | I | ł | I |
| 9 FA 9 LF | +31.95 | +44-75 | | 47.35 | I | ļ | I | t | I |
| R.W. | 85.75 | 47.65 | 6, ⁷ | | - | | | | |
| | + 0 • 0 | +e•4 | ` ∔ I | | | | | | |
| REGRESSION | | | | | | 0 * 1 * * * * 0 1 1 1 * * * * ノノ ご * - |) 1 ** | * * ((1 | ** |
| • * • | 121.40 128 25 | I | I | 1 | 404•00 +66.53 | 20.020 115.15 | 01°C11 | 74.76 | |
| L L | | | | | | | | + | 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 |
| 5 M • U | 40.04 | 1 | 1 | I | | | | | |
| | 412.00 | | | | +10.1 | <u>+</u> 20.6 | F0.6+ | +4.13 | +00.00 |

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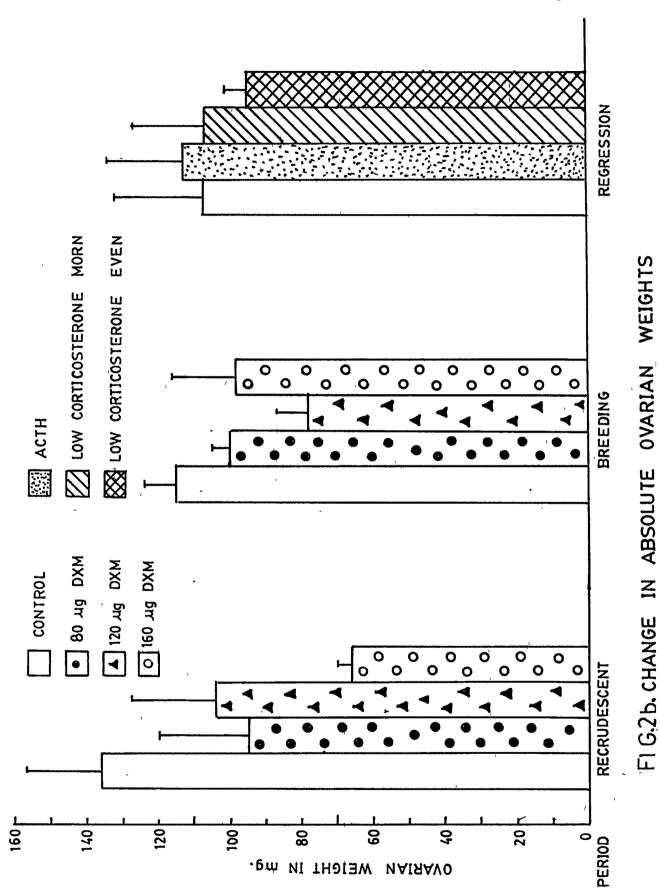


| TABLE-2b | | SEASONAL ALT (OVARY) (mg4 MENTAL PIGEC | SEASONAL ALTERATIONS IN ABSOLUTE (OVARY) (mg+S.D.; mg/100 gm BODY MENTAL PIGEONS, C.LIVIA | IN ABSOLUTE 100 gm BODY IA | | ATIVE WE Lasod.) | AND RELATIVE WEIGHT OF FEMALE GONAD WEIGHT <u>*</u> S.D.) OF NORMAL AND EXPERI- | EMALE GONA AND EXPER | |
|------------------------|----------------------------|--|---|---|----------------------------------|------------------------|--|--------------------------------|----------------------------------|
| REPRODUCTIVE PHASES | NORMAL | Boyg | DEXAMETHASONE 120µg 16 | oug | ACTH 0.5 I.U. | 1µgM | соги 1удЕ | CORTICOSTERONE JµgM | 正 Jµ侶王 |
| RECRUDE SCENT A.W. | 136.05 421.07 | 94.76* <u>+</u> 26.22 | 104.54 [@] <u>+</u> 24.71 | 66.06 +4.92 | ۱ * | ı | ı | ı | ł |
| R.W. | 50.38 +5.00 | 38°41+ | 40°46 * +8•0 | 27 .43 +9.0 | * | | | | |
| BREEDING A.W. | 115.74 <u>4</u> 9.93 | 99.85* <u>+</u> 16.84 | *1 | 77.58 ^{***} 97.82 [*] 10.26 <u>+</u> 19.06 | I | I | ı | ı | 1 |
| R.W. | 49.25 +10.0 | 42.48 48.6 | 35•26 <u>+</u> 7•0 | 46.40 <u>*</u> 8.0 | | | | | |
| REGRESSION A.W. | 107 •46 <u>+</u> 25 •86 | I | ı | I | 113.40 <u>+</u> 21.13 | 95•23 <u>+</u> 6•00 | 107.5 <u>+</u> 20.12 | 113.7 <u>+</u> 28.84 | 109.45 +13.36 |
| R.W. | 42•58 <u>⊀</u> 8•0 | ı | 1 | 1 | 44.76, 48.011 | 51.19 ±5.0 | 39 • 68* + 6•4 | 44 . 06 <u>+</u> 9.0 | 36 . 12 <u>+</u> 12.00 |
| | + P< 0.01 M - MORNING | е С. н С. | <pre>< 0.02</pre> | *: P<0.05 G A.W | *** P< 0.0005 ABSOLUTE WEIGHT | 0.0005 Weight | R.W RI | RELATIVE WEIGHT | TGHT |

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| TABLE-3 | •• | NAL ALTEN S.D.; mu NS, C.LI | SEASONAL ALTERATIONS IN ABSOLUTE AND RELATIVE WEIGHT (mg ± S.D.; mg/100 gm BODY WEIGHT ± S.D.) OF NORMAL A PIGEONS, C.LIVIA | I ABSOLUTE 30DY WEIGH | T <u>+</u> S.D.) | TIVE WEIG OF NORMA | ND | THYROID EXPERIMENTAL | .ı | |
|---|----------------|-----------------------------------|---|-----------------------------|----------------------|-----------------------|----------------------|-------------------------|------------|-----|
| REPRODUCTIVE PHASES | NORMAL | Boug | DEXAMETHASONE 120µg 16 | sone 160µg | ACTH 0.5 I.U. | 1JugM | CORT 1µgE | CORTICOSTERONE 348M | NE JygE | 1 1 |
| RECRUDESCENT A.W. | 10.12 | 14.30+ | 14 . 88* | 12 . 48 ⁴ | ı | ı | , | 1 | ż | |
| | <u>+</u> 2.20 | 4.00 | +3.10 | +2.89 | | | | | | |
| R.W. | 3.71 | 6.195 | ** 6.17*** | 5.63*** | | | | | | |
| | 40 . 68 | +0.60 | +0.50 | +0.50 | | | | | | |
| BREEDING A.W. | 8.78 | 12.85 * | 12.72** | 11.65 ⁰ | 1 | 1 | , | I | ı | |
| | +2.35 | <u>*</u> 2.56 | | | | | | | | |
| R.W. | 3.49 | 5.32 | 5.47 | 5.08 | 1 | I | | | | |
| | 40.84 | 1.01 | ÷1.00 | 11°11 | | | | | | |
| REGRESSION A.W. | 19.27 | I | ı | I | 13.36* | 12.68* | 22 . 88 | 19.18 | 19.16 | • |
| | 4.81 | | | | +3.28 | • • | · · · | 41.83 | +5.47 | |
| R.W. | 7.26 | t | I | ł | 5.13 | 4.96 | 9.67** | 7.55 | 8.016 | |
| | +0.76 | | | | 41.01 | | ~ 1 | 06°0 7 | +0.80 | |
| a de la companya de l | + PC0.01 | 0 | P_0.02 *P | 0•05** | *P 0.05 ** P 20.005 | | *** P< 0.0005 | | | |
| | M - Morning | ы | - Evening | A.B. Ab | A.B. Absolute Weight | | R.W. Relative weight | ive weigh | ۲ د | |

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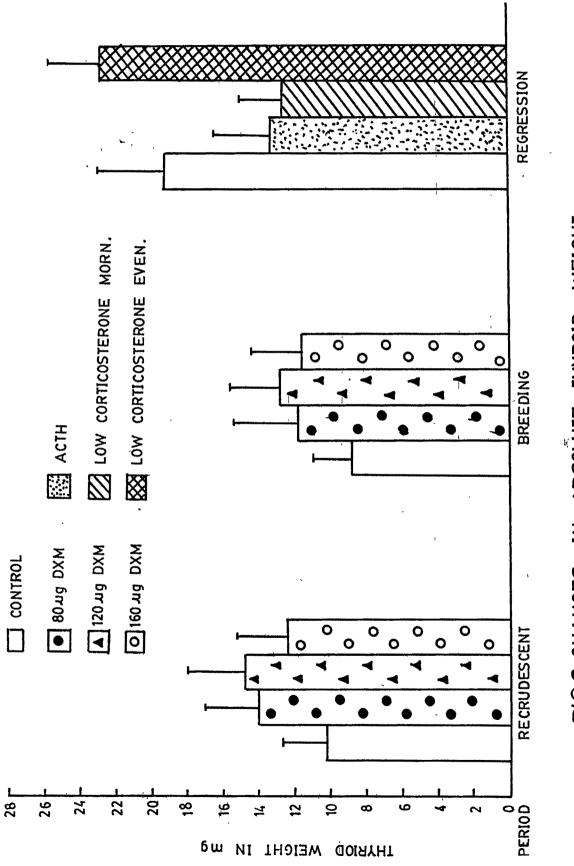


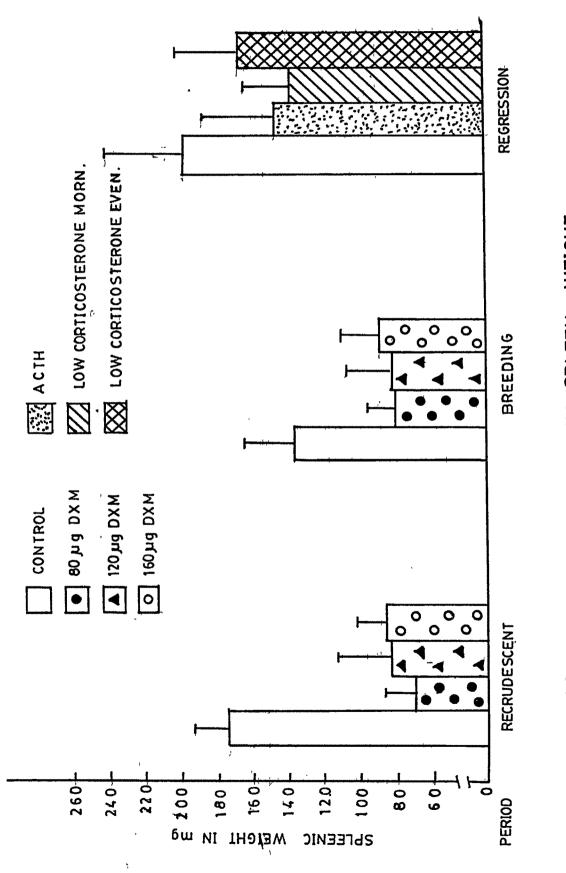
FIG.3. CHANGES IN ABSOLUTE THYROID WEIGHT

| TABLE-4 | | SEASONAL ALTERATIONS IN (mg ± S.D.; mg/100gm BOD MENTAL PIGEONS, C. LIVIA | S IN ABSOLUTE m BODY WEIGHT LIVIA | | AND RELATIVE WEIGHT OF SPLEEN ± S.D.) OF NORMAL AND EXPERI- | SPLEEN | |
|-------------------------|--|---|---|-----------------------------------|---|--|--------------------------|
| REPRODUCTI VE PHASES | NORMAL | 80µ£ | DEXAMETHASONE 120µg | E 160µE | ACTH .0.5 I.U. | CORTICOSTERONE 1µgM 1µgE | TERONE 1µgE |
| RECRUDESCENT A.W. | 173.98 | 70.56*** | 85 . 18 *** | 81.95 | 1 | ₿ | l |
| R.W. | ±18.97 63.84 ±5.13 | ±18•17 30•57 ±6•00 | +28•56 35•32 +7•00 | <u>+</u> 20•09 36•98 +4•32 | Ĩ | ł | ı |
| BREEDING A.W. | 136 . 18 +28 . 9 | 79.66 +16.4 | 81 . 18 +27.3 | 89.46 +30.38 | ı | ı | I |
| R.W. | .+ .+ | 33.02*** +2.30 | | 39.03 +6.02 | ı | ł | I |
| REGRESSION A.W. | 196.26 | - 1 | I | I | 146.02* | 137.28* | 166.63* |
| R.W. | <u>+</u> 25.66 73.99 <u>+</u> 7.00 | I | ı | ı | +41 •28 56.16 +3.0 | + 26.74 53.72 + 10.1 | ±36.10 70.48 ±6.40 |
| | * P<0.05 M - MORNING | ** P<0.005 ★E - EVENING | ** NG | * P<0.0005 A.W ABSOLUTE WEIGHT | | R.W RELATIVE WEIGHT | EIGHT |

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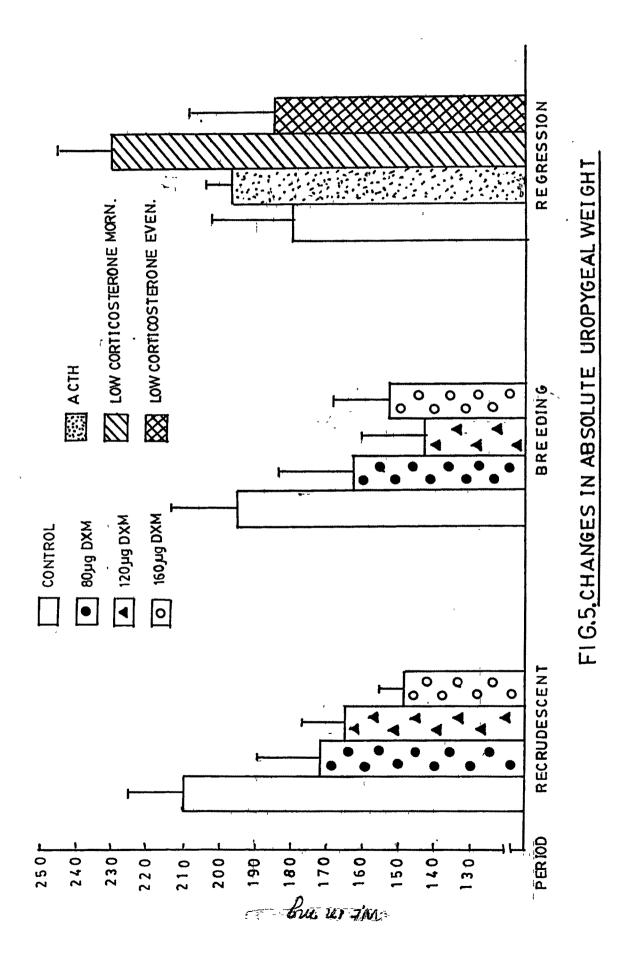
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| REPRODUCTIVE NORMAL DEXAMETHASONE ACTH A | TABLE-5 | | SEASONAL ALTERATIONS (gm ± S.D.; mg/100gm PIGEONS, C. LIVIA | ATIONS IN 100gm BO. VIA | IN ABSOLUTE BODY WEIGHT | | TIVE WEIG OF NORMAL | AND RELATIVE WEIGHT OF UROPNGIUM - S.D.) OF NORMAL AND EXPERIMENTAL | PKGIUM RIMENTAL | |
|---|-------------------------|---------------|---|-------------------------------|----------------------------|---|------------------------|--|------------------------|---------------|
| NT 81.47 64.92 44.98° 49.38° $-$ W. 81.47 64.92 44.958 ± 11.85 $-$ W. 29.89 25.12° $18.65^{\ast\ast}$ 22.28° $-$ W. 101.6 $57.28^{\ast\ast\ast}$ $44.26^{\ast\ast\ast}$ $54.16^{\ast\ast\ast\ast}$ $-$ W. 40.46 23.74° $19.03^{\ast\ast\ast\ast}$ $23.63^{\ast\ast\ast\ast\ast}$ $-$ W. 40.46 23.74° $19.03^{\ast\ast\ast\ast}$ $23.63^{\ast\ast\ast\ast\ast}$ $-$ W. 79.66 $ 216.00^{\circ}$ W. 79.66 $ 23.63^{\circ}$ $-$ W. 79.66 $ 216.00^{\circ}$ W. 20.03° <td>REPRODUCTI VE PHASES</td> <td>NORMAL</td> <td></td> <td>EXAMETHAS</td> <td>ONE 160µg</td> <td>ACTH 0.5 I.U.</td> <td></td> <td>CORT 1ygE</td> <td>CORTICOSTERONE JugM</td> <td>压 318日</td> | REPRODUCTI VE PHASES | NORMAL | | EXAMETHAS | ONE 160µg | ACTH 0.5 I.U. | | CORT 1ygE | CORTICOSTERONE JugM | 压 318日 |
| ± 24.46 ± 16.78 ± 9.58 ± 11.85 W. 29.89 25.12^* 18.65^{***} 22.28^{\odot} ± 4.00 ± 2.00 ± 1.90 ± 4.00 W. 101.6 57.28^{***} 44.26^{***} 54.16^{***} W. ± 10.16 57.28^{***} ± 11.55 ± 11.55 W. ± 10.46 23.74^* $\pm 19.03^{***}$ 23.63^{***} W. ± 6.00 ± 2.00 ± 2.00 $\pm 3.60^{\circ}$ W. 79.66 $ 99.12^{*}$ W. 79.66 $ 38.12$ W. 79.66 $ 38.12$ W. 79.60 $ -$ W. 79.60 $ \pm 14.00$ ± 7.00 ± 2.00 ± 7.00 ± 7.00 W. 79.66 $ -$ W. 79.00 ± 2.00 ± 7.00 ± 7.00 | RECRUDESCENT A.W. | 81.47 | 64.92 | 44.98 ⁰ | 49.38 ⁰ | ł | 1 | | | 3 |
| W. 29.89 25.12* 18.65** 22.28 ^a - ± 4.00 ± 2.00 ± 1.90 ± 4.00 W. 101.6 57.28*** 441.26 *** 54.16 *** - ± 15.27 ± 11.45 ± 12.34 ± 11.55 W. 40.46 23.74 *** 19.03 *** 23.63 *** ± 6.00 ± 2.00 ± 2.00 ± 3.90 W. 79.66 99.12* W. 79.66 38.12 ± 14.00 W. 30.03 38.12 ± 3.00 ± 7.001 @ $P < 0.02$ * $P < 0.05$ ** $P < 0.00$ | | 424.46 | +16.78 | | T | | | | | |
| $\begin{array}{cccccccccccccccccccccccccccccccccccc$ | R.W. | 29,89 | 25.12* | | | ł | 1 | I | I | ł |
| W. 101.6 57.28 *** 44.26 *** 54.16 *** - ± 15.27 ± 11.45 ± 12.34 ± 11.55 W. ± 0.46 23.74 ± 19.03 *** 23.63 *** ± 6.00 ± 2.00 ± 2.00 ± 3.90 ± 16.00 ± 2.00 ± 2.00 ± 3.90 W. 79.66 99.12 ± 14.00 W. 50.03 58.12 ± 36.00 ± 70.01 @ P < 0.02 * P < 0.05 ** P < 0.00 | | ±4.00 | <u>+</u> 2.00 | | | | | | | |
| W. 101.6 57.28 44.26 54.16 5 ± 15.27 ± 11.45 ± 12.34 ± 11.55 W. 40.46 23.74 19.03 ± 23.63 ± 11.55 W. ± 6.00 ± 2.00 ± 2.00 ± 3.90 W. 79.66 - 2.00 ± 2.00 ± 3.90 ± 14.00 W. 30.03 38.12 ± 3.00 ± 79.01 @ P ≤ 0.02 $\pm P_{\leq 0.05}$ $\pm P_{\leq 0.00}$ | BREEDING | | : | : | : | | | | | |
| $\begin{array}{rrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrr$ | A. W. | 101.6 | 57 . 28 | * 44.26 | * 54.16 | | ı | ł | 1 | I |
| W. 40.46 23.74 *** 19.03 *** 23.63 *** ± 6.00 ± 2.00 ± 2.00 ± 3.90 W. 79.66 99.12 ± 14.00 W. 30.03 38.12 ± 3.00 ± 7.00 $\mathbb{R} > 0.03$ + P < 0.01 @ P < 0.02 * P < 0.05 ** P < 0.00 | | 15.27 | +11.45 | +12.34 | +11.55 | | | | | |
| $\begin{array}{rrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrr$ | R.W. | 40.46 | 23.74 | * 19.03 | * 23.63** | * | | | | |
| W. 79.66 99.12* ±14.00 W. 30.03 ±16.0 ±3.00 + P∠0.01 @ P∠0.02 * P∠0.05 ** P∠0.0 | | + 6.00 | +2.00 | +2.00 | +3.90 | | | | | |
| ±14.00 ±14.00 ±30.03 38.12 ±3.00 + P<0.01 @ P<0.02 * P<0.05 ** P<0.0 | REGRESSION | 70 66 | 1 | I | ł | * ° ° ° ° ° ° ° ° ° ° ° ° ° ° ° ° ° ° ° | 105 144 | 105 00 + | 64, 87 | 68 <u>4</u> 5 |
| 30.03 38.12 ±3.00 + P<0.01 @ P<0.02 * P<0.05 ** P< 0.00 | • | +14.00 | I | I | I | +16.0 | +15.2 | +11.1 | +15.8 | +11.23 |
| ±4.00 @ P∠0.02 * P<0.05 ** P< 0.00 | R.W. | 30.03 | ł | ı | I | 38.12 | 41.15 | 44.80 | 25.53 | 28.64 |
| @ P<0.02 * P<0.05 | | +3.00 | | | | <u>+</u> 4.00 | <u>+</u> 2•00 | 4.00 | + 2•0 | +2.00 |
| | | + P<0.0 | 0 | * | P< 0.05 | ** P< 0. | | *** P< 0.0005 | 5 | |
| M - MORNING E - EVENING A.W ABSOLUTE WEIGHT | | M - MORN | 더 | EVENING | A.W A | BSÒLUTE W. | | R.W RELATIVE WEIGHT | ATIVE WE | IGHT |

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Changes in experimental animals :

Dexamethasone (dxm) treatment during the active phases (i.e. recrudescence and breeding) induced fall in adrenal weights With all the three doses employed (i.e. 80 µg, 120 µg, and 160 µg). Injection of ACTH or corticosterone during the regression phase brought about an increase in adrenal weight. Similarly, the gonads too exhibited a par Jallel set of changes with dxm treatment inducing lowered weights, and ACTH/Corticostorone treatment inducing increased weights. However, the female gonad (ovary) did not reveal significant increase in weight in response to ACTH/Corticosterone as was the case with the male gonad (festis).

Thyroid gland recorded an increase in weight in dxm treated birds during the active phases with the weights tending to be in the range of those recorded for normal birds during the regression phase. ACTH and LCM during the regression phase brought about decreased thyroid weights, a change which was characteristic of the normal birds during the breeding phase. However, LCE and HCM as well as HCE failed to evoke any significant response.

Unlike the response of the adrenals, gonads and the thyroid, the spleen depicted a different response under

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experimental conditions. Whereas the normal birds had increased spleen weights during regression and decreased weights during recrudescence and breeding, both dxm treatment during breeding and ACTH/Corticosterone administration during regression brought about significant reduction in spleen weight. Uropygium or preen gland was found to depict a decrement in weight in dxm treated birds during the active phases ACTH and LCM during the regression phase on the other hand brought about increment in uropygial weight. However, HCM and HCE failed to bring about any significant alterations.

DISCUSSION

Though both antagonistic and synergistic relationships between adrenal and gonads have been reported in birds, all the available data on mammals and the majority of those available on birds tend to favour a parallel adrenal-gonad interrelationship (Riddle, 1923; Legait and Legait,1959; Fromme-Bouman,1962; Bhattacharya and Ghosh,1965; Höhn <u>et al</u>, 1965; Ramachandran and Patel, 1986). The gravimetric changes recorded for adrenal and gonads during the course of the present study clearly indicate a parallel adrenal-gonad axis in tropical blue rock pigeons as both the adrenal and gonad weights registered their maximum during the reproductively active months. This is further confirmed by the minimal weights recorded by both the organs during the post-breeding months. Histological observations have also shown increased adrenocortical activity when the monds were gametogenically active, and reduced cortical activity when the gonads were quiscent (Chapter - III). Earlier works from this laboratory on the pineal-gonad axis of tropical wild and domestic pigeons, had also suggested a parallel adrenal-gonad axis (Ramachandran <u>et al.</u>, 1985; Ramachandran and Patel, 1985), and the present study has helped confirm the relationship between adrenal and gonads inferred thereat.

Further validity to this concept is provided by the results of the present study on experimental manipulation of adrenals and the resultant effect on gonads. Accordingly, adrenal suppression by dxm during the reproductively active phases has led to decreased adrenal weight paralleled by shrunken, regressed gonads in the experimental birds, a picture corresponding to the reproductively quiscent phase in normal birds. Again, administration of ACTH or corticosterone in the non-breeding phase could induce significant increase in adrenal and gonad weights, changes characteristic of normal birds during the recrudescent and breeding periods.

Being a trophic hormone, the increased adrenal weight recorded under the influence of ACTH is understandable and has been reported to occur in other species of

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birds as well (Riddle et al., 1924; Poll, 1925; Garren et al., 1961; Zarrow et al., 1962). However the increase in adrenal weight shown by birds administered with corticosterone is enigmatic and needs some alternate explanation which is elusive at this juncture. ACTH and LCM both induced increase in testicular weight, and the testicular weights recorded being very much similar to that of recrudescent phase thus suggesting a state of gonadal activation (in an otherwise inactive phase) characteristic of recrudescence. However, LCE, HCM and HCE all failed to evoke any increase in testicular weight with the latter regimes tending to decrease the testicular weight further. The results emphasize the dose and time specifications involved in the response of tissues and organs to hormones and as such the need to take into consideration these aspects while executing experiments and interpreting the data involving hormonal manipulations. Apparently, the male gonads of tropical Indian feral blue rock pigeons seem to exhibit diurnal sensitivity/refractoriness in responding to an optimum titer of corticosteroid as denoted by the gonadal enlargement obtained by LCM and no effect with LCE. Once again the negative response of the gonads to HCM and HCE indicate the inhibitory influence of supraoptimal levels of hormones even at the sensitive phase. Time and dose specific responses of avian gonads to circulating levels of corticosterone $\langle \widehat{f} \rangle$

in terms of a circannual refractoriness has been suggested. Hamner (1968) claims a circadian component timing the refractory period in <u>Carpodacus mexicanus</u>. The studies of Benoit <u>et al</u>. (1956) in ducks and that of Lofts (1962) in Quelea suggest the occurrence of a photorefractory period and the involvement of a circadian based mechanism in determining the duration of photorefractivity. Further, a more positive involvement of the testis in the underlying endocrinological mechanisms of the refractory period is also provided by the recent work of Murton <u>et al</u>. (1970).

ACTH or corticosterone affecting the weight of gonads or other reproductive structures has also been reported; as Khan <u>et al.</u> (1977) recorded an increase in oviducal weight along with significant increase in plasma and ovarian bestrogens in the spiny tailed lizard. Similarly, high doses of corticosterone have been reported to inhibit ovulation while low doses have proved to be stimulatory when injected at a critical period during the ovulatory cycle of hen (Rzasa <u>et al.</u>, 1983). Further, Flickinger (1966) reported that ACTH induced increase in steroid output led to a dose related linear reduction in the size of the oviduct and testes in fowl. Rise in levels of corticosteroids prior to ovulation in birds has been reported by andEtches (1979) Etches and Simons (1979). However, injections

of corticosteroid at this phase would lead to treméndous level increase in endogenous corticosteroid, which in the light of present observations could be considered to cause adversory effects. Pertinently, massive doses of ACTH or corticosterone have been known to inhibit or cause premature ovulation in hen (Tienhoven, 1961). A casual observation of table 1 shows that the seasonal changes in ovarian weights are less dramatic and even non-significant than those of the testicular weights. ACTH injection increased the ovarian weight (closer to that characteristic of breeding phase) though statistically non-significant, while LCM and LCE did not increase the weight at all. However, HCM did show a positive response identical to that obtained with ACTH and hence highlights another aspect involving sex difference in optimal dose specificity. Apparently the female gonads too, though depicting a morning responsiveness needs a higher titre of corticosteroid or a slightly longer duration of sensitization. Malendowicz (1979) in his study on adult Wistar rats reported sex difference in adrenocortical structure and function; the relative and absolute adrengal weight being lower in males than in females and the cortical region of females being wider than in males. The reports of Zaleska and Krystyna (1979) who observed higher levels of corticosteroids in females than in males of lab mice also provides added credence.

Thyroid-gonad axis in birds has also been looked into by many investigators and have yielded results indicating both parallel and inverse relationships. (Thapliyal and Pandha, 1967 a, b; Jallageas and Assenmacher, 1973, 1974; Chandola and Thapliyal, 1974; Oishi and Konishi, 1978; Jallageas et al., 1978). Effect of functional status of thyroid on gonads in mammals also has been reported (Cook, 1960; Hara, 1963; Cadariu et al., 1976; Jha, 1986). Parallel relationship of thyroid with gonads in domestic pigeons and inverse relationship of the same in wild pigeons have been observed by Ramachandran and Patel (1986) and Ramachandran et al. (1987) respectively. Inverse thyroid-gonad and adrenal-thyroid relationships could be inferred from the present study. Heralding of breeding activities is marked by a fall in thyroid weight and cessation of breeding activities by an increase in weight. In experimental birds, dxm suppression of adrenals during the breeding phase exhibited increased thyroid weight along with regressed gonads. Reverse set of changes was the feature in ACTH and LCM treated birds during the non-breeding phase. Neither LCE nor HCM or HCE could decrease the thyroid weight, once again emphasizing the dose specificities as well as diurnal sensitivity of avian tissues to hormonal fluctuations and the inherent endocrine interactions involved in seasonal modulation of reproductive activities.

Though there are only few reports regarding the adrenal-spleen axis, an inverse adrenal-spleen weight relationships has been inferred by Jonathan (1978) in matured and pregnant bats. Even the studies on developing chicks have indicated suppression of bursa with increasing age and increasing activity of adrenal gland thus interpreting an negative correlation between the adrenal gland and bursa (Glick, 1970). Several authors have demonstrated bursal regression in presence of cortisone or corticosterone or ACTH (Glick, 1957a, b; 1959; 1960; 1967, 1972; Zarrow et al., 1961; Bellamy and Leonard, 1964; Sato and Glick, 1964, 1970; Dieter and Breintenbach, 1970, 1971). Further, Glick and Dreesen (1967) based on their studies on birds reported that species with small bursa possessed adrenal glands that were significantly larger than that of birds with large bursa. An obvious inhibitory influence of adrenal on lymphoid organs can be inferred from these reports, and the present observations of inverse seasonal relationship between adrenal and spleen weights in normal pigeons and the reduced spleen weights in dxm treated as well as ACTH/Corticosterone administered birds tend to strengthen the above inference. In the light of the purported inverse adrenal-spleen relationship, though the reduced spleen weight recorded in ACTH/Corticosterone injected birds is understandable, the reduction in weight shown by dxm treated birds along with fall in adrenal

weights may appear contradictory. However, an explanation can easily be sought in the fact that dxm being a synthetic corticosteroid could mimic the actions of corticosteroids inspite of suppression of the adrenals and hence spleen is not capable of distinguishing between the native corticosteroid molecule and the synthetic analog. This might also indicate the existance of two types of Corticosteroid receptors, one more specific which could respond to the native molecules only and the other non-specific or less specific which could respond to both the native molecules and the analog as in the case of spleen. Though the spleen responded to both low as well as high doses of corticosterone in a dose related fashion, the reduction in weight obtained by the morning dose was more significant than that obtained by the evening dose thus suggesting a differential diurnal sensitivity of the spleen as well.

Uropygium or the preen gland was found to exhibit maximum weight during the breeding season and least weight during the non-breeding season, and in the light of the fact that this gland is a target organ for gonadal steroids, the present results are justifiable. Gupta and Maiti (1983) reported that gonadal steroids (estradiol dipropionate) increased the mitotic frequency and cell differentiation in uropygial gland resulting in increased lipid droplets.

Decreased glandular weight during the non-breeding phase could be due to lowered levels of gonadal steroids thereby resulting in reduced lipid formation. In adrenal suppressed birds along with gonadal involution, uropygeal weight also decreased. Experimentally induced hyperadrenalism by ACTH/ Corticosterone administration increased Gropygeal weight along with gonadal enlargement. All these results give validity to the concept of Uropygium as the target organ for gonadal steroids. Again, high doses of corticosterone which failed to bring about gonadal enlargement also failed to increase the weight of the Gropygeal gland.

SUMMARY

Gravimetric analysis of gonads, adrenals, thyroid, spleen and uropygium has been carried out during normal seasonal cyclicity as well as under conditions of adrenal manipulation in <u>Columba livia</u>. Increased weights of adrenals and uropygium have been recorded along with increased gonadal weights during the breeding phases, while decreased weights of thyroid and spleen were recorded concurrently. Both, the normal non-breeding phase as well as adrenal suppression by dxm during the breeding phase induced a reverse set of changes, while ACTH/corticosterone treatment in the nonbreeding phase induced changes akin to that of recrudescent and breeding phases. These changes are taken to suggest the operation of a parallel adrenal-gonad axis as well as an inverse relationship between steroids and uropygium.